

# MILITARY MEDICINE

## CHANGES IN PULSE CHARACTER AND MENTAL STATUS ARE LATE RESPONSES TO CENTRAL HYPOVOLEMIA

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### ABSTRACT

**Objective.** Manual assessment of radial arterial pulse character remains an important determinant of physiological status in military and civilian casualties. This study hypothesized that changes in radial pulse character and mental status (presyncopal symptoms) in humans occur only during the later stages of progressive reductions in central blood volume in close association with systolic blood pressure (SBP). **Methods.** Pulse character (i.e., normal, weak, absent), estimated stroke volume (SV), SBP, and diastolic blood pressure were measured continuously during baseline supine rest and during progressive reductions of central blood volume to cardiovascular collapse with application of lower body negative pressure (LBNP) in 19 healthy human volunteer subjects. **Results.** LBNP resulted in a progressive reduction in SV. At early stages of LBNP, both radial pulse character and SBP were well-maintained. Although both radial pulse character and SBP decreased with subsequent increases in LBNP, these changes occurred only after an approximate 55% reduction in SV and were associated with the onset of presyncopal symptoms. Changes in mental status did not occur until the point of cardiovascular collapse. **Conclusions.** In this model of progressive central hypovolemia secondary to application of LBNP in humans, radial pulse character score decreased in concert and was highly correlated with decreases in SBP. In support of our hypothesis, changes in pulse character, SBP, and mental status occurred only after significant reductions in SV, suggesting that these standard vital signs may not be early indicators of central hypovolemia. **Key words:** hemorrhagic shock; lower body negative pressure; cardiovascular collapse.

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### INTRODUCTION

Hemorrhagic shock remains a leading cause of death worldwide in both civilian and combat trauma.<sup>1–3</sup> Consequently, providing capabilities for timely assessment of blood loss could significantly improve patient outcome by prioritizing attention to those at risk of circulatory collapse and signaling the need for earlier life-saving intervention(s). Current triage practices utilize physiologic data readily obtainable at the site of injury, which are assumed to provide a real-time snapshot of the patient's condition. Specifically, a low systolic blood pressure (SBP < 90 mmHg) and / or mental status (Glasgow Coma Score motor component [GCSm] < 6) have been considered good indicators for mortality and / or the need for life saving intervention.<sup>4–7</sup>

Unfortunately, the tools available to assist providers in assessment of blood loss are subjective and based on nontrended discrete manual sampling of vital signs. Perhaps more limiting is that changes in the standard vital signs obtained from current medical monitors (e.g., arterial oxygen saturation, heart rate, respiration rate) can be significantly delayed because of physiologic compensatory mechanisms, thus potentially delaying intervention and increasing the risk of poor outcome. This notion is best supported by the frequent anecdotal reporting by physicians and medics of trauma patients who enter a hospital with normal vital signs but subsequently decompensate and die. An analysis of data from 1300 patients from our civilian trauma patient database indicates that this subset of patients can represent a significant proportion of trauma (unpublished data).

Because of current logistical constraints and the impracticality of placement of automated diagnostic equipment into far-forward tactical and multiple-casualty incident environments, prehospital medical personnel experience greater limitations in triage decision making and evacuation priorities for patients. Patient measurements that do not require monitoring

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equipment may be the only way to evaluate casualties in such environments. For this reason, manual assessment of radial pulse remains an important determinant of status in military and civilian casualties as a possible tool to estimate SBP.<sup>8</sup> Despite a controversy about the value of pulse palpability in patients for predicting blood pressure and clinical outcome,<sup>9–11</sup> the concept of using pulse character as a hemodynamic diagnostic tool is attractive to military medical personnel operating in austere environments and to civilian providers responding to mass casualty situations.

We previously analyzed data from our prehospital vital signs database<sup>12</sup> and reported that worsening radial pulse character and GCSm was associated with patient mortality and indicated the need for an intervention just as well as standard vital signs recorded from current automated medical monitors.<sup>13</sup> It is unclear, however, whether the association between patient outcome and pulse character and mental status, like that between patient outcome and other standard vital signs such as SBP,<sup>14</sup> reflects a physiological outcome that appears only after the patient is in crisis rather than a compensatory response that might prove helpful for earlier diagnosis and intervention. The purpose of this investigation was to test the hypothesis that the onset of a weak or absent radial pulse character and changing mental symptoms in humans occurs during the later stages of progressive reductions in central blood volume in close association with SBP.

## METHODS

### Subjects

Demographic data from the nineteen healthy non-smoking subjects recruited to participate in this study are shown in Table 1. A complete medical history and physical examination were obtained on each of the potential subjects. In addition, female subjects underwent an initial urine test prior to experimentation to ensure that they were not pregnant. Subjects maintained their normal sleep pattern, refrained from exercise, and abstained from caffeine and other autonomic stimulants, such as prescription or nonprescription drugs, for at least 24 hours prior to each experimental protocol. During an orientation session that preceded each experiment, all subjects received a verbal briefing and a written description of all procedures and risks associated with the experiments, and were made

TABLE 1. Subject Demographics. Data are means  $\pm$  SD.

	Males (n = 12)	Females (n = 7)	Total (n = 19)
Age (years)	32 $\pm$ 10	26 $\pm$ 7	29 $\pm$ 9
Height (cm)	181 $\pm$ 6	165 $\pm$ 11	175 $\pm$ 11
Weight (kg)	92 $\pm$ 12	61 $\pm$ 14	80 $\pm$ 20

familiar with the laboratory, protocol, and procedures. Experimental procedures and protocols were reviewed and approved by the institutional review board for the use of human subjects at the Brooke Army Medical Center at Fort Sam Houston, Texas. Each subject gave written, informed voluntary consent to participate in the experiments.

### Model of Central Hypovolemia

Lower body negative pressure (LBNP) was used in the present investigation as an experimental tool to simulate loss of central blood volume (e.g., hemorrhage) in humans.<sup>15</sup> With the use of a neoprene skirt designed to form an airtight seal between the subject and the chamber, the application of negative pressure to the lower body with the subject in a supine position results in a redistribution of blood away from the upper body (head and heart) to the lower extremities and abdomen (Fig. 1A). Thus, this model provides a unique method of investigating conditions of controlled progressive, experimentally induced hypovolemic hypotension. Although equivalence between the magnitude of negative pressure applied and the magnitude of actual blood loss has not been absolutely determined, our previous review of both human and animal data reveal ranges of effective blood loss (or fluid displacement) caused by LBNP.<sup>15,16</sup> As an example, we have proposed that negative pressures greater than 40 mmHg induce hemodynamic responses that are equivalent to those resulting from blood loss approximating 1000 mL.<sup>15,16</sup>

### Experimental Design

All subjects were instrumented with an infrared finger photoplethysmograph (Finometer® Blood Pressure Monitor; TNO-TPD Biomedical Instrumentation, Amsterdam, The Netherlands) to record estimates of beat-to-beat arterial pressures<sup>17–20</sup> The Finometer blood pressure cuff was placed on the middle finger of the left hand, which, in turn, was laid at heart level. Mean arterial pressure (MAP) was calculated by dividing the sum of SBP and twice diastolic blood pressure by three. An electrocardiogram (ECG) was recorded to provide pulse rate.

Beat-to-beat stroke volume (SV) was measured non-invasively using thoracic electrical bioimpedance with an HIC-2000 Bio-Electric Impedance Cardiograph (Bio-Impedance Technology, Chapel Hill, NC). The thoracic electrical bioimpedance technique is based on the resistance changes in the thorax to a low-intensity (4 mA), high-frequency (70 kHz) alternating current applied to the thorax by two surface electrodes placed at the root of the neck and two surface electrodes placed at the xiphoid process at the midaxillary line. Ventricular SV was determined with the partly empirical

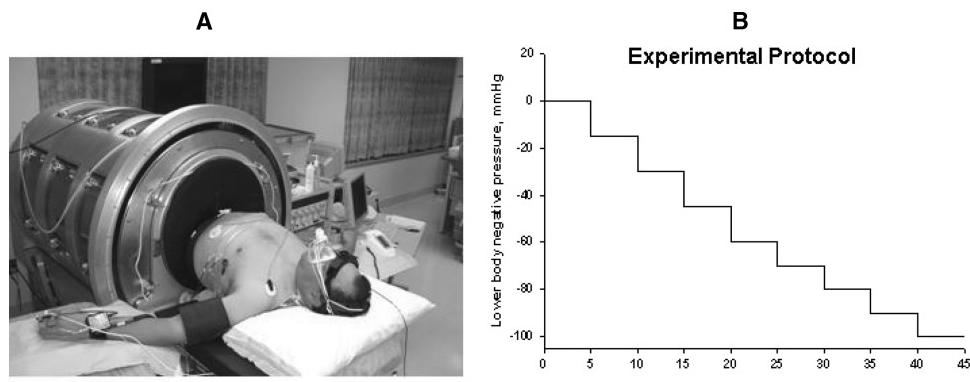


FIGURE 1. Subject in the lower body negative pressure (LBNP) device (A) and the LBNP experimental protocol (B).

formula:  $SV \text{ (in ml)} = \rho \times (L/Z_0)^2 \times LVET \times (dZ/dt)$ , where  $\rho$  (in ohm/cm) is the blood resistivity, a constant of 135 ohms/cm *in vivo*; L (in cm) is the mean distance between the inner-band electrodes (front and back);  $Z_0$  (in ohms) is the average thoracic background impedance; LVET (in seconds) is the left ventricular ejection time; and  $(dZ/dt)$  is the maximum height of the  $dZ/dt$  peak measured from the zero line.<sup>21</sup> Correlation coefficients of 0.70–0.93 have been reported in SV measurements simultaneously made with thoracic electrical bioimpedance and thermodilution techniques.<sup>22</sup>

Each subject underwent exposure to an LBNP protocol designed to test his or her tolerance to experimentally induced hypotensive hypovolemia. The LBNP protocol consisted of a five-minute baseline period (0 mmHg) followed by five minutes of chamber decompression to -15, -30, -45, and -60 mmHg, and additional increments of -10 mmHg every five minutes until the onset of cardiovascular collapse or the completion of five minutes at -100 mmHg (Fig. 1B).

Cardiovascular collapse was defined by one or a combination of the following criteria: 1) a precipitous fall in SBP >15 mmHg; 2) a sudden decrease in HR > 15 beats per minute (bpm); 3) progressive diminution of SBP below 70 mmHg; and/or 4) voluntary subject termination due to onset of presyncopal symptoms (e.g., alterations in vision, sweating, nausea, or dizziness). Upon cardiovascular collapse, LBNP was halted and

the pressure within the chamber immediately returned to atmospheric pressure (0 mmHg).

At each LBNP level, pulse character in the radial artery was evaluated by a physician (AB) and was classified as "normal" (a strong pulse that was easy to palpate; score = 2), "weak" (palpable pulse but difficult to find; score = 1); and "absent" (no pulse found; score = 0). The examiner was blinded to the subject's medical history and fitness level and arrived at the experimental site immediately before baseline data collection with the subject already in the LBNP chamber in the supine resting condition. While the examiner was familiar with the experimental protocol, he was blinded to the experimental measurements (e.g., blood pressures, heart rate, etc.) as they were being taken throughout the experiment. There was no verbalization of the current LBNP level by experimental personnel during the experiment, although subjects were asked if they were experiencing presyncopal symptoms at each LBNP level. It was impossible to completely blind the examiner to the ongoing procedures, as he could hear changes in the sound of the LBNP device and could also hear questions asked of the subject.

## Outcome Measures

The primary outcome measures were pulse character and mental status. Secondary outcome measures were blood pressures, SV, and heart rate.

TABLE 2. Cardiovascular Measurements Before (Baseline) and at the Point at which Statistical Significance Was Achieved during LBNP.

Parameter	Baseline Value	Significant Value	% LBNP Tolerance where Statistically Different from Baseline	p-value
Systolic blood pressure (mmHg)	131 ± 11	124 ± 10	60	0.001
Diastolic blood pressure (mmHg)	77 ± 7	80 ± 7	60	0.039
Mean arterial pressure (mmHg)	98 ± 8	94 ± 7	80	0.001
Heart rate (bpm)	65 ± 8	79 ± 12	60	<0.001
Pulse pressure (mmHg)	54 ± 9	49 ± 9	40	0.015
Stroke volume (ml)	132 ± 33	98 ± 31	40	<0.001
Radial pulse character (au)	2.0 ± 0.0	1.7 ± 0.4	80	0.023

## Statistical Analysis

Subjects reached cardiovascular collapse (i.e., maximal LBNP tolerance) at different absolute LBNP levels based on their individual physiological responses. Because these responses are the same at cardiovascular collapse independent of the LBNP level at which an individual subject reaches this point,<sup>23</sup> we chose to normalize each individual's data by reapportioning their responses to equal fractions between 0% LBNP tolerance (baseline) and 100% LBNP tolerance, the level at which the LBNP protocol was terminated as a result of impending cardiovascular collapse (presyncope). This approach allowed us to consider the data from all subjects relative to their maximum capacity for LBNP tolerance.

Cardiovascular variables were averaged over the last three minutes of each LBNP level. A one-way (LBNP level) randomized block (subjects) analysis of variance

for repeated measures was used for comparison of outcome variables. If statistical differences were found, Bonferroni-corrected comparisons with baseline measurements were performed to determine the first level of LBNP that could be distinguished statistically from baseline. Regression analysis was used as appropriate to correlate changes in variables. All data are presented as mean  $\pm$  standard deviation (SD), and exact p-values are presented for all comparisons.

## RESULTS

The mean time to cardiovascular collapse was  $1738 \pm 285$  seconds. Of these 19 subjects, four experienced cardiovascular collapse during  $-45$  mmHg, seven during  $-60$  mmHg, six during  $-70$  mmHg, and two during  $-80$  mmHg.

Cardiovascular responses during LBNP are shown in Figure 2. LBNP produced a progressive reduction

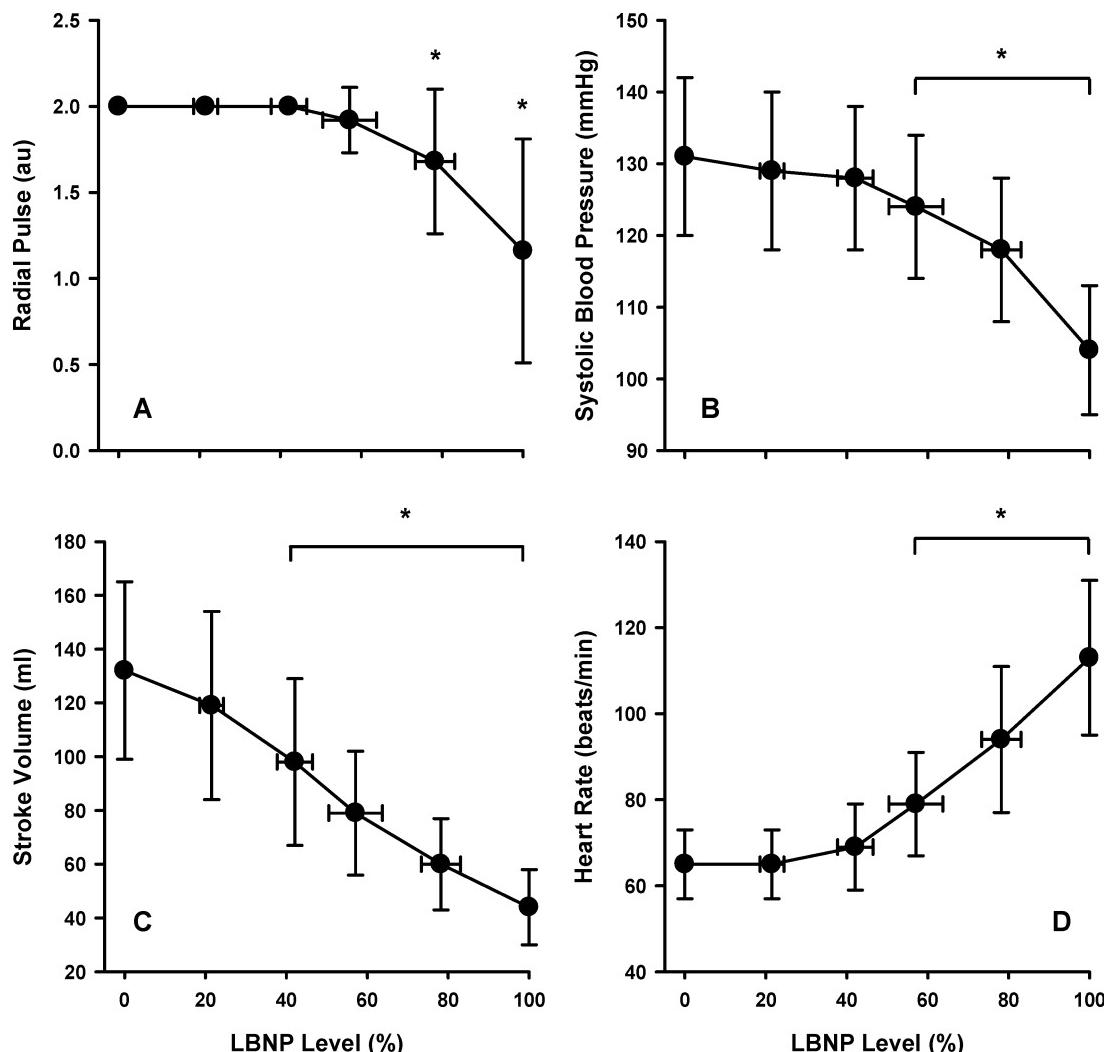


FIGURE 2. Radial pulse character (A), systolic blood pressure (B), stroke volume (C), and heart rate (D) throughout the lower body negative pressure (LBNP) protocol. Due to intersubject variation in LBNP time to cardiovascular collapse, responses to LBNP were reapportioned to equal fractions between 0% maximum LBNP (baseline) and 100% maximum LBNP, the level where cardiovascular collapse occurred and the LBNP protocol was stopped. \* $p \leq 0.02$  compared with baseline value. Data are means  $\pm$  SD.

in central blood volume, as indicated by a linear decrease in SV. Despite this progressive central hypovolemia, SBP and radial pulse character were maintained until the late stages of the experiment and deteriorated abruptly after application of 60% and 80% LBNP, respectively (Table 1, Fig. 2). At 80% maximal LBNP level, the radial pulse character had a rating of "weak" (score = 1) in 5 of the 19 subjects and remained "normal" (score = 2) in the remaining 14 subjects. At the same 80% LBNP level, SV decreased to  $60 \pm 17$  mL, a 55% reduction from baseline (Fig. 2). HR increased from baseline levels progressively and significantly after application of 60% maximal LBNP.

A second-order regression analysis of the relationship between changes in radial pulse character and

changes in SV revealed an amalgamated correlation coefficient ( $R^2$ ) of 0.98 (Fig. 3, top). In contrast, changes in radial pulse character were linearly associated with the changes in SBP ( $R^2 = 0.99$ ; Fig. 3, bottom).

Throughout the LBNP protocol, subjects reported no signs or symptoms of presyncope (i.e., alterations in vision, sweating, nausea, or dizziness). Such events occurred only at the point at which blood pressure decreased suddenly and LBNP was terminated. At this point, SBP and DBP were  $84 \pm 14$  and  $59 \pm 15$  mmHg, SV was  $50 \pm 18$  mL, and HR was  $108 \pm 28$  bpm (values obtained by averaging over the last 10 heart beats before LBNP termination). The associated radial pulse character measurements were 2 ("normal") in six subjects, 1 ("weak") in eight subjects, and 0 ("absent") in five subjects.

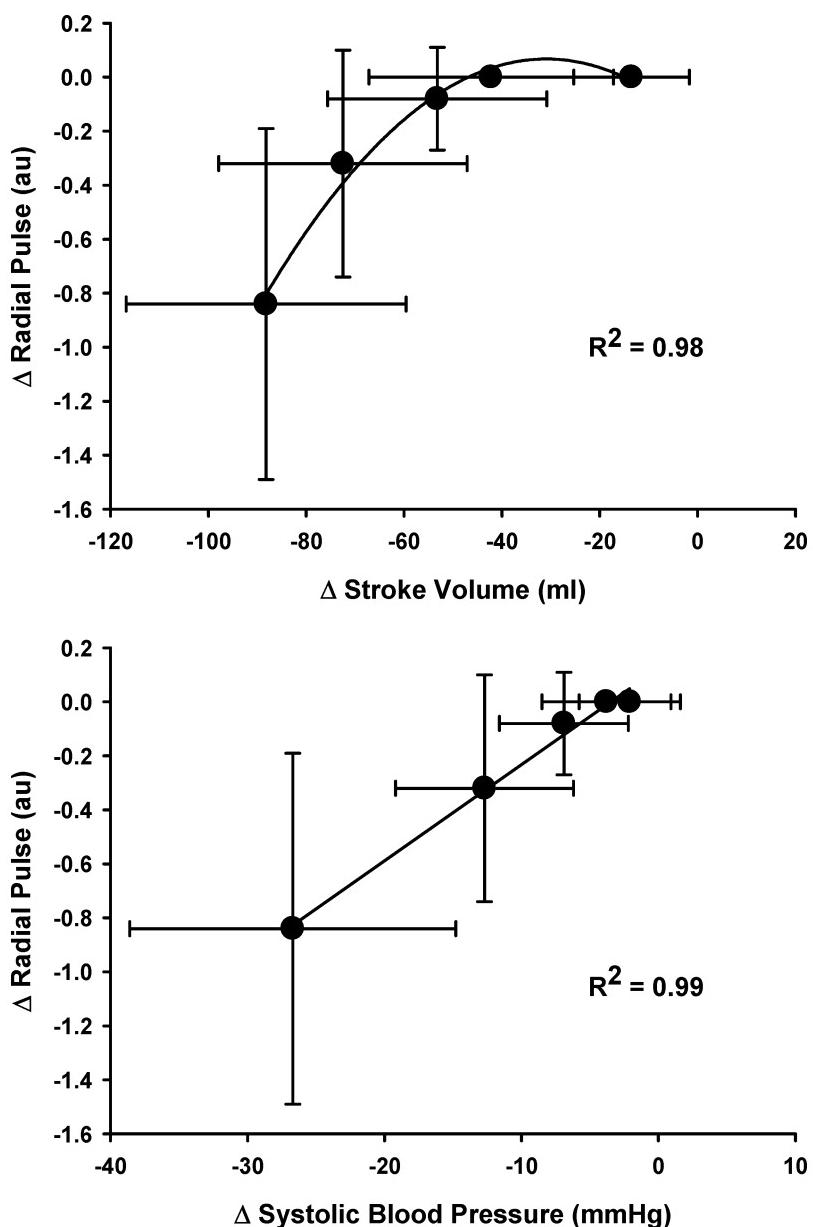


FIGURE 3. Changes in radial pulse character plotted against changes in stroke volume (top) and systolic blood pressure (bottom) during the lower body negative pressure protocol. Data are means  $\pm$  SD.

## DISCUSSION

Because the ability to obtain blood pressure measurements in an austere environment is often limited by time constraints, equipment availability, and noisy conditions, the use of palpable radial pulse to estimate SBP has been advocated.<sup>8,12</sup> Contradictory interpretations about the actual sequence and value of radial pulse palpability in patients for predicting blood pressure and clinical outcome exist, with some physicians continuing to support the use of pulse character in triage decision making, while others report its limited value.<sup>9–11</sup> Further, a difference of opinion also exists concerning both the ability to obtain a pulse and the value of observations of pulse weakening in the arteries commonly used for predicting SBP during circulatory collapse.<sup>24–26</sup> Thus, the diagnostic value of palpable pulse characteristics in trauma patients remains controversial.

In the present investigation, we provide the first direct evidence that changes in human radial pulse character caused by experimentally controlled reductions in central blood volume similar to those experienced during hemorrhage<sup>15</sup> are direct linear reflections of reduced SBP. More importantly, our experimental model of central hypovolemia provides a method of trended continuous sampling that substantiates that weakening pulse character reflects a clinical outcome that manifests itself at a time when it may be too late to apply effective intervention or triage.

Previously, the ability of casualties to follow commands (GCSm) and their SBPs revealed the strongest association with critical injury when utilizing physiologic variables for trauma triage in a multiple casualty incident.<sup>4–6,13</sup> In a recent study,<sup>12</sup> we analyzed medical records of 274 trauma patients taken to a Level 1 trauma center and reported that an abnormal radial pulse was associated with significantly lower SBP and higher mortality than those of patients with a normal pulse. Subsequently, we analyzed data from 381 prehospital trauma patient records collected during helicopter transport by EMS personnel and found that the receiver operating character (ROC) area calculated from radial pulse character, and motor scores obtained only by physical examination were equal to the ROC area obtained from currently available diagnostic equipment (0.97).<sup>13</sup> The results of the present investigation support these earlier observations of the close relationship between radial pulse character and GCSm obtained from physical examination and outcomes. Our present data extend our understanding of this relationship by demonstrating that weak or absent radial pulse and presyncopal symptoms do not appear until just prior to the point of circulatory decompensation and cardiovascular collapse. Although this information may be clinically useful, it would be more desirable to use a triage tool that provides information before this point is reached. In this sense, if the provider's actions are

driven by detrimental changes in pulse character or GCSm alone, the chances of administering timely life-saving interventions or triage decision would be vastly diminished.

The use of arterial pulse character as a guide to diagnose and treat various injuries by medical providers is not a new idea. The concept of estimating SBP in trauma patients based on palpable pulses was formalized in the Advanced Trauma Life Support (ATLS) course manual printed in 1985.<sup>8</sup> The manual formalized the relationship that SBP was more than 80 mmHg if the radial pulse was palpable in addition to the carotid and femoral pulses. Teaching the association between palpable pulses and SBP was subsequently discontinued after the printing of the 1985 course manual because of the absence of data to support such a relationship. Using our LBNP model, we have been able to provide the first data that support a linear relationship between SBP and radial pulse characteristics in humans undergoing progressive hypovolemic hypotension leading to circulatory collapse. Our data are consistent with the ATLS guidelines in that, on average, a palpable but weak radial pulse was associated with an average SBP of just greater than 80 mmHg.

## Limitations

Our study has a few limitations worthy of discussion. First, our laboratory model of progressive central hypovolemia provided a unique opportunity to collect physiologic data on human volunteers, but we recognize that cardiovascular responses to experimentally induced central hypovolemia may be different when compared with responses to actual severe hemorrhage. Although LBNP induces fluid redistribution from the upper to lower body rather than actual blood loss, previous experiments have shown that many of the physiological responses to LBNP are similar to those of actual volume loss up to as much as 1000 mL.<sup>15</sup> Of note, the fact that LBNP is not hemorrhage may also be seen as a benefit, inasmuch as we were able to assess the relationship between radial artery pulse character and reduced central volume without confounding factors, such as tissue injury and painful stimuli that might affect autonomic nervous system activity. Therefore, our results may be the first to strictly reflect the relationship between reduced central blood volume and pulse character in human subjects. Next, measurements of pulse character were obtained by a single provider. Although we recognize that this decreases experimental variability, we acknowledge that this approach does not represent the true prehospital environment where various discrete samplings occur at different time points and are carried out by multiple providers. Further, while we made every attempt to blind the physician to the LBNP level and the standard vital signs at the time of his measurement, it was impossible to completely

abrogate the possibility of measurement bias. Third, the use of normal, healthy subjects could have biased the results. In most previous studies, subjects had underlying pathophysiology which may influence the pulse character. Finally, measuring SV with bioimpedance cardiology is an indirect method. Although we observed the expected progressive, linear reductions of SV with LBNP,<sup>14,15</sup> we stress that the actual volume of fluid displaced was not determined.

## CONCLUSION

In this investigation, we have employed continuous vital sign monitoring in a human model of progressive hypovolemic hypotension to demonstrate that: 1) a weak or absent radial pulse and presyncopal symptoms do not appear until just prior to the point of circulatory decompensation and cardiovascular collapse and 2) worsening radial pulse character is associated with decreases in SBP. Our findings do not dismiss the importance of assessing radial pulse character and mental status as relevant and effective clinical tools for patient status. However, our results emphasize that a detrimental change in pulse character reflects a critical point at which the caregiver may have little time to provide an effective intervention that could beneficially influence patient outcome.

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